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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

Johns-Manville

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SEP 30 1985
OFFICE OF PESTICIDES AND TOXIC SUBSTANCES

SEP 25 1985

U.S. EPA, REGION V
WASTE MANAGEMENT DIVISION
HAZARDOUS WASTE ENFORCEMENT BRANCH

MEMORANDUM

SUBJECT: Response to Region V Request for Enforcement Support, Johns-Manville Site

FROM: Elizabeth A. Dutrow, Chemist
Field Studies Branch
Exposure Evaluation Division (TS-798)

TO: Rodney Gaither, RPM
Hazardous Waste Enforcement Branch/Region V

The following memo discusses the Final Remedial Investigation Report, Volumes I and II, on the Johns-Manville Waukegan Disposal Site. As you recall, the original sampling and analysis protocol and the Quality Assurance Plan were prepared with support from the Exposure Evaluation Division (EED). To facilitate the review, the main author of the protocol was again called upon to review the final report. Each of your requests are answered below.

1) Evaluate data on airborne asbestos.

Upon review of the documents, it is evident that the original protocols and Quality Assurance Plan have been reproduced in the "Consent Order," which requires Johns-Manville to carry out the Remedial Investigation. The air sampling program, conducted by Eric Chatfield, is identical to the plan within the Consent Order. No fault is found with this activity.

The airborne levels detected are consistent with Chatfield's previously reported ambient levels. Additionally, a recent study conducted by EED displayed similar ambient levels (Evaluation of Asbestos Abatement Techniques, Phase I). Hence, the conclusion by Chatfield that the levels of the Manville Site are not elevated is reasonable.

2) Evaluate the need for further remedial action at the site, based on the asbestos test.

The Johns-Manville levels appear to be consistent with the reported airborne data available. Note, however, should the site or asbestos characteristics undergo any sort of change

which would result in an increase of friability in the asbestos, materials, additional remedial action may be necessary. Periodic sampling would detect any changes in the airborne levels. Is periodic sampling a form of "further remedial action?" If so, then periodic sampling would be appropriate.

3) Compare the airborne asbestos test to other reliable airborne asbestos tests that have been done before.

As stated previously, the design and Quality Assurance Plan are well-developed. The execution of the work followed the plan, and the analyst has a good reputation. Additionally, the airborne asbestos levels are low.

4) Recommend how the airborne asbestos problem at this site can be better described in the Endangerment Assessment.

5) Recommend how the asbestos problem in water samples can be better described in the Endangerment Asssessment.

Further detail is necessary to adequately the issues. ? How is the current description deficient?

6) Recommend a suitable way to address the issue on health and safety of the public on drinking liquids containing asbestos.

Please refer to the attached pages from the National Research Council's Study "Asbestiform Fibers: Nonoccupational Health Risk" (1984). The attached pages (119-123) discuss studies examining the consumption of water containing millions of fibers per liter. These levels are similar to those reported in the technical memorandum M-1, "Asbestos Analysis of Water Samples by Electron Microscopy." Since the results) -? the NRC study are unclear,) I suggest that you contact Dr. James Millette. Dr. Millette works for EPA in Cincinnati (within your Region). His phone number is FTS-684-7462. He may also provide some further assistance to you as an additional reviewer. (Dr. Millette has examined the issue of asbestos in water supplies in this country.)

? ← ↓ No longer there (EPA) ↓

Attachment

Tom Powers FTS 684 7550
or
Bill Keane

Nat'l. Research Council's 1984
"Asbestiform Fibers: Nanoccupational Health Risks

Summary

Persons residing in areas in Turkey where asbestiform fibers are present in the environment and persons living in the same household as workers exposed to asbestos develop mesothelioma at a rate in excess of that for the general population. The evidence is based primarily on clinical observations and on case-control studies that do not permit generalization. It seems likely that these mesotheliomas arise from respiratory exposure to asbestiform fibers.

EPIDEMIOLOGICAL STUDIES OF EFFECTS RESULTING FROM THE INGESTION OF ASBESTOS IN DRINKING WATER

Epidemiological studies of the effects of asbestos in drinking water in six geographical areas of the United States and Canada have been extensively reviewed and critiqued (Marsh, 1983; Workshop on Ingested Asbestos, 1983). In all these studies, a possible excess incidence of gastrointestinal (GI) cancers was evaluated as were morbidity or mortality rates for some other cancers. In addition, the National Research Council's Safe Drinking Water Committee addressed this problem and estimated the risk of excess GI cancers associated with ingesting asbestos in drinking water (National Research Council, 1983a).

Tables 5-1, 5-2, and 5-3 summarize the characteristics and results of the various studies. Duration of exposure ranged from as little as 20 years (in Duluth[6]) to more than 50 years (in Quebec); asbestos concentrations ranged from less than detectable limits to 1,300 x 10^6 fibers/liter. Except for Duluth, where taconite mine tailings were dumped into Lake Superior, the subjects were exposed to chrysotile from natural sources (in Quebec, the San Francisco Bay area, and Puget Sound) or from asbestos-cement pipes (in Utah and Connecticut).

The studies did not indicate consistent excesses of cancer. In Duluth, no consistent type of cancer occurred in excess among residents (Levy et al., 1976; Mason et al., 1974; Sigurdson et al., 1981). In Quebec, cancer mortality was evaluated in relation to asbestos in municipal water supplies. In the first study (Wigle, 1977), 22 municipalities were grouped into three categories based on level of asbestos in water supplies. In a more extensive study (Toft et al., 1981), mortality rates for two cities with high exposure (>100 x 10^6 fibers/liter) were compared with 52 low exposure cities (<5 x 10^6 fibers/liter). Some excess cancers in males that were noted in the two studies were attributed to probable occupational exposure. In Connecticut, tumor registry data indicated that there was no association

[6]The particles in Lake Superior were mostly acicular cleavage fragments rather than asbestiform fibers (T. Zoltai, personal communication, 1983). See also Langer et al., 1979.

TABLE 5-1. Characteristics of Asbestos Exposures from Drinking Water in Different Study Populations[a]

Location of Study	Exposure Characteristics: Type of Asbestos	No. of Fibers per Liter (Range)	Size of Population Exposed	Maximum Duration of Exposure (Years)
Duluth	Amphibole[b]	1-30 x 10^6	100,000	15-20
Connecticut	Chrysotile	BDL[c]-0.7 x 10^6	576,800	23-44
Quebec	Chrysotile	1.1-1,300 x 10^6	420,000	50
Bay Area, California	Chrysotile	0.025-36 x 10^6	3,000,000	40
Utah	Chrysotile	NA[d]	24,000	20-30
Puget Sound	Chrysotile	7.3-206.5 x 10^6	200,000	40

[a]From Marsh, 1983.
[b]Most of these particles were probably acicular crystals rather than asbestiform fibers (T. Zoltai, University of Minnesota, personal communication, 1983). Langer et al. (1979) referred to the particles as amphibole gangue minerals and discussed the uncertainties in determining whether they are asbestiform.
[c]BDL = below detectable limit.
[d]NA = not available.

between asbestos risk scores and GI tumor incidence (Harrington et al., 1978; Meigs et al., 1980). In San Francisco, there were inconsistent excesses of some cancers (Conforti et al., 1981; Kanarek et al., 1980; Tarter, 1981). In Puget Sound, a proportional incidence analysis comparing length of residence suggested an excess for some GI cancers (Polissar et al., 1982).

All of the epidemiological studies had limitations. Perhaps the most serious were the substantial problems in classifying exposure because population data rather than individual data were used. Errors in classification will tend to weaken any true associations that may exist between asbestos in drinking water and health effects. Given the difficulty of determining individual exposure, results of these epidemiological studies cannot be taken as strong evidence about the extent to which ingestion of drinking water containing asbestiform fibers might increase the risk of GI cancer. The NRC Safe Drinking Water

TABLE 5-2. Summary of Studies of Gastrointestinal Cancer in Relation to Ingested Asbestos by Cancer Site[a]

Location	Association of GI Cancer with Asbestos, by Site[b] (ICD 7th Revision Codes)										References
	All Sites Combined (150-159)	Esophagus (150)	Stomach (151)	Small Intestine (152)	Colon (153)	Rectum (154)	Biliary Passages/ liver (155-156A)	Gall Bladder (155.1)	Pancreas (157)	Perito-neum (158)	
Duluth	(++)	(+-)	(++)	NS	(00)	(++)	(00)	NS	(0+)	NS	Mason et al., 1974
Duluth	(--)	(00)	(+0)	(00)	(--)	(00)	(00)	(00)	(++)	(00)	Levy et al., 1976
Duluth	(00)	(00)	(00)	(00)	(00)	(00)	(00)	(00)	(0+)	(00)	Sigurdson et al., 1976
Connecticut	NS	NS	(00)	NS	(00)	(00)	NS	NS	NS	NS	Harrington et al., 1978
Conneticut	NS	NS	(00)	NS	(00)	(00)	NS	NS	(+0)	NS	Meigs et al., 1980
Quebec	(00)	(00)	(+0)	NS	(00)	(00)	NS	NS	(0+)	NS	Wigle, 1977
Quebec	(+0)	(00)	(+0)	NS	(00)	(00)	NS	NS	(00)	NS	Toft et al., 1981
Bay Area, Calif.	(++)	(0+)	(++)	(00)	(00)	(00)	(00)	(0+)	(0+)	(++)	Kanarek et al., 1980
Bay Area, Calif.	(++)	(++)	(++)	(00)	(+0)	(00)	(00)	(00)	(++)	(0+)	Conforti et al., 1981
Bay Area, Calif.	(++)	NS	NS	NS	NS	NS	NS	NS	NS	NS	Tarter, 1981
Utah	NS	NS	(00)	(00)	(0-)	(00)	NS	(0+)	(00)	(00)	Sadler et al., in press
Puget Sound	(00)	NS	(00)	NS	(--)	NS	NS	NS	NS	NS	Severson, 1979
Puget Sound	NS	(00)	(00)	(++)	(00)	(00)	(00)	(00)	(00)	(00)	Polissar et al., 1982

[a]From Marsh, 1983.
[b](Male, female) association with ingested asbestos.
+, positive; 0, no association; - negative; NS, not studied.

TABLE 5-3. Summary of Studies of Risk from Cancer Other Than Gastrointestinal Cancer in Relation to Ingested Asbestos, by Cancer Site[a]

Location	Association of Cancer Other Than GI with Asbestos, by Site[b] (ICD 7th Revision Codes)									References
	Buccal Cavity and Pharynx (140-148)	Bronchus, Trachea and Lung (162,163)	Pleura (162.2)	Prostate (177) (males only)	Kidney (180)	Bladder (181)	Brain/ CNS[c] (193)	Thyroid (194)	Leukemia, Aleukemia (204)	
Duluth	NS	(+0)	NS	NS	NS	NS	(00)	NS	(00)	Mason et al., 1974
Duluth	NS	NS	NS	NS	NS	NS	NS	NS	NS	Levy et al., 1976
Duluth	NS	(00)	NS	NS	NS	NS	NS	NS	NS	Sigurdson et al., 1976
Connecticut	NS	NS	NS	NS	NS	NS	NS	NS	NS	Harrington et al., 1978
Connecticut	NS	(00)	NS	NS	(00)	(00)	NS	NS	NS	Meigs et al., 1980
Quebec	(00)	(+0)	NS	0	(00)	(00)	(00)	NS	(00)	Wigle, 1977
Quebec	(00)	(+0)	NS	0	(00)	(00)	(00)	NS	(00)	Toft et al., 1981
Bay Area, Calif.	NS	(+0)	(0+)	0	(0+)	(00)	(00)	(00)	(00)	Kanarek et al., 1980
Bay Area, Calif.	NS	(00)	(0+)	+	(00)	(00)	(00)	(00)	(00)	Conforti et al., 1981
Bay Area, Calif.	NS	NS	NS	NS	NS	NS	NS	NS	NS	Tarter, 1981
Utah	NS	NS	NS	NS	(+0)	NS	NS	NS	(+0)	Sadler et al., 1981
Puget Sound	NS	NS	NS	NS	(00)	NS	NS	NS	NS	Severson, 1979
Puget Sound	(00)	(00)	NS	+	(00)	(00)	(+-)	(++)	(+-)	Polissar et al., 1982

[a]From Marsh, 1983.
[b](Male, female) association with ingested asbestos.
+, positive; 0, no association; -, negative; NS, not studied.
[c]CNS = central nervous system.

Committee (1983a), using a variety of assumptions, estimated the excess risk of GI cancers that might be expected from ingestion of asbestos-containing drinking water and concluded that their risk estimates are consistent with the results of the epidemiological drinking water studies considered.

OCCUPATIONAL EPIDEMIOLOGICAL STUDIES--METHODOLOGICAL CONSIDERATIONS

Evaluation of potential health effects from nonoccupational exposure to asbestiform fibers depends primarily on results of epidemiological studies of occupational groups. Most of the analyses have involved cohort[7] studies of workers exposed to asbestos of various types and in a variety of industries and occupations. Much information has been obtained from these studies. However, they also suffer from limitations common to many epidemiological studies and from some additional problems related to determining dose (exposure) and response (health end point, such as death from a specific cause). Despite the limitations of individual studies, the committee finds that, when all the studies are considered, exposure to asbestos increases the risk of developing lung cancer, mesothelioma, asbestosis, and possibly other cancers.

To quantify health risks from an exposure, it is necessary to obtain dose-response data, but exposure measurements are particularly difficult to obtain. Because of the long latency period for asbestos-associated diseases, investigators have found it necessary to try to reconstruct past exposures. Techniques of measurement vary from place to place and over time (Acheson and Gardner, 1980; Dement et al., 1983a). For example, fiber counts obtained by light microscope in various industrial settings may need to be multiplied by a factor varying from 2 to 8 to obtain a true count of fibers longer than 5 μm.

Typically, a cumulative dose measurement is used. This does not take into account the time lapsed since last exposure nor does it distinguish between short exposures of high intensity and long exposures to low dust concentrations. In addition, a cumulative dose measurement does not change when exposure ceases. Variability in these exposure-related

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[7]The two major types of epidemiological studies are cohort studies and case-comparison studies. In a cohort study, a group with certain defined characteristics of exposure is selected and followed to determine the number of members reaching a particular end point, such as death, by a specified time. The group is called a cohort. In its purest form, the analysis of a cohort study depends entirely on within-cohort comparisons, and the results may be presented as arrays of morbidity or mortality rates or by a large variety of other expressions of association or correlation. A cohort might comprise two major groups, differentiated by their exposure experience. However, in occupational studies, especially of cancer, the rate of occurrence of death or disease in the group is often compared with the rate in some

(continued)